made; Ookubo et al, Journal Pharmaceutical Sciences (November 1992), 81(11), 1139-1140. However, such compounds still result in an intolerable amount of aluminum accumulation in renal failure patients. It is also known to use calcium compounds having pool solubility at pH 6-9, eg calcium carbonate, hydroxide, oxide and/or sulphate in a medicinal form resistant to gastric juices. However, it is known that, for example, with calcium carbonate, a large dosage is required because of its relatively low in vivo capacity for phosphate removal, such large dosages also being difficult to administer. This can cause further complications associated with high calcium intake. It has also been proposed (WO-A-92/01458) to control serum phosphate levels in patients suffering from or predisposed to hyperphosphataemia by contacting ingested phosphate with an oxy-iron compound selected from ferric oxides, oxy-hydroxides and hydroxides. Similarly, Spengler et al, Nephrol. Dial. Transplant. (1996), 11, 808-812, suggests treatment of hyperphosphataemia with a complex of iron (III) oxide-hydroxide modified dextran. However, in the tests conducted, extremely high dosage amounts to animals were given. Moreover, many inorganic preparations are efficient phosphate binders only over a limited pH range, especially an acid pH range of about 3-5. Such current phosphate binders effective at pH3 would not necessarily bind as effectively at higher pH, eg $\geq$ 7, which obtain in the lower tract, eg duodenum and below, and where at least some of the binding of phosphate may take place. Moreover, particularly alkaline binders could buffer the stomach pH up to a high level at which they would not have a phosphate binding capacity.--

See the attached Appendix for the changes made to the specification.